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Stirling, J. W.

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EYE SYMPTOMS OF INTRACRANIAL ORIGIN.

BY

J. W. STIRLING, M.B. (EDIN.), MONTREAL.

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Dr. F. G. Finley

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CERTAIN EYE SYMPTOMS OF INTRACRANIAL ORIGIN.

By J. W. STIRLING, M.B. (EDIN.), MONTREAL.

(Read before the Canadian Medical Association, at Ottawa, September, 1888.)

The subject of my paper, "Certain Eye Symptoms of Intracranial Origin," has to do with that well-known and perhaps rather threadbare subject, "The Abnormal Limitations of the Field of Vision,"—i.e., scotomata, hemianopsia, etc.,—and also the limitations of the field of color perception. These limitations, in the light of some recent investigations by Willbrand and others, are, indeed, of clinical importance to the general practitioner as well as specialist in enabling him to localize certain cerebral lesions.

I will endeavor, within the limits of a short paper, to give a *resumé* of these investigations, illustrating them by a few cases which have come under my notice. I trust it may not be out of place if I at first run over the course and ultimate destination of the optic nerve fibres. Proceeding backwards from the eye, the optic nerves decussate partially at the chiasma, after which, taking the name of optic tracts, they continue backwards, bending round the pedunculus cerebri. Fibres from the right tract supply the right side of both retinæ, and those from the left tract the left side. The macula lutea of each eye is connected, it is at present assumed, with both cerebral hemispheres. But, to return to the optic tracts themselves, they arise :

(1) By fibres from the grey substance of the optic thalamus and the anterior corpora quadrigemina, the corpora geniculata forming ganglia intercalated in the course of certain of the fibres.

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(2) Fibres come from the tegmentum of the crus.

(3) Also of marked import are certain fibres which pass directly through the medulla oblongata without the intervention of grey matter, forming the so-called spinal root of the optic tract, and explaining the pupillary reaction on the stimulation of the retina by light.

(4) A broad band of fibres passes from the tract to the psycho-optic centre at the apex of the occipital lobe, called the optic radiation of Gratiolet.

Other connections of the optic tracts with the cerebral hemispheres exist, far too numerous to mention in detail here ; indeed Gratiolet affirms that the optic tracts are connected directly with every part of the cerebral hemispheres from the frontal to the occipital lobes. Some fibres are ganglionic, arising from the basal ganglia, and others cortical in origin—both uniting to form the optic tracts.

Lastly, I may mention as of importance that certain fibres cross in the corpus callosum from the motor areas of the opposite cerebral hemisphere, enter the outer capsule, and join the tract directly.

Commencing in front at the optic nerve, I will now, passing backwards, give examples of lesions in various parts of the visual tract. Lesions of the optic nerve associated with monocular blindness and dilatation of the pupil are common, but I may be permitted to cite as an example of a certain class of these cases by no means very common one which I lately had under treatment :—

J. P., aged 45, seen March 12th, complaining of loss of sight in his left eye. About New Year he slipped on the ice and fell, striking his forehead just over the supra-orbital ridge. Was at that time treated for fracture of the skull. Three weeks later the vision of the left eye began to get impaired, and has since steadily got worse until now it is abolished. Examination revealed a deep depression over the left orbit, at the junction of the inner and middle thirds, extending from the edge of the orbit one inch upwards ; deep palpation can also discover a depression in the roof of the orbit, extending backwards in the direction of

the optic foramen. Ophthalmoscopic examination showed haziness of the margins of the disc, two or three vessels hidden, and atrophic pallor beginning.

These cases of fracture of the orbital roof extend into the optic foramen, set up a retrobulbar neuritis, followed by atrophy of the optic nerve, or at other times act by pressure caused by exudation or hemorrhage.

Coming now to the chiasma as the seat of lesions causing a limitation of the field of vision, I saw a case, during my house-surgeon days at the Royal Infirmary in Edinburgh, of the rare condition of paralysis of the inner halves of both retinae—*i.e.*, blindness of the temporal sides of the field.

W., aged 23, was seen by Dr. Berry. Although the patient was almost moribund, still the defect of both fields of vision to the temporal side was clearly observable. The patient died a few days later, when the post-mortem revealed a tumor the size of a small hen's egg occupying the region of the pituitary body and involving nearly the whole chiasma.

I have at present another case of evident lesion at the chiasma.

W. J., aged 42, came to me in February complaining of complete loss of vision in the left eye entirely, and partial loss in the right eye. Sight began to fail in the left eye two years ago, and for the past six months has been completely abolished. The right eye began to fail eighteen months ago, but for the past eight months there has been no change. No history of syphilis, but a marked alcoholic one. Left eye, no perception of light. Right eye, complete blindness to outer side of field—*i.e.*, paralysis of nasal side of retina. Inner portion of field amblyopic. Counts fingers at twelve feet. Pupil, right eye active; left eye motionless, except on stimulation of right eye.

Here the lesion undoubtedly affects the anterior part of the chiasma, since the nasal portions of both retinae are paralyzed, and likely, also, the adjacent portion of the left optic tract is affected. Ophthalmoscope showed both discs pale and atrophic.

Cases of homonymous hemianopsia or blindness of the same areas of both retinae are invariably caused by lesions posterior to the chiasma. Those occurring in front of the anterior cor-

pora quadrigemina—*i.e.*, in the optic tract—being distinguished from those occurring behind them in the occipital lobes by the loss of the pupillary reflex.

The reaction of the pupil on stimulation of the retina by light being abolished in the former case, but retained in the latter, together with absence of dilatation, although there is no psychical perception of light.

This is easily understood by considering the anatomical conditions, for it is in the region of the anterior corpora quadrigemina that the tract gives off its spinal root, through which the reflex travels to the third nerve, which innervates the sphincter pupillæ. The centres are in the medulla, and are coupled, so that both pupils react, although only one retina be stimulated.

The experiments of Curschmann, Haab and others have conclusively proved the existence of a unilateral innervation centre for corresponding portions of both retinæ.

The lesions in post-mortem sections have been found to occupy portions of the cerebral occipital lobes, viz., the first, second and third, and the cuneus.

Mank excised the occipital lobes of a dog, causing paralysis of the same sides of both retinæ—*i.e.*, blindness to the opposite side in the field of vision. On excising the occipital lobes of both hemispheres, although the animal was totally blind, yet the pupils reacted readily to light.

Schaefer has found that on excising all of the occipital lobes except a layer of the lower surface in the monkey, the eye was entirely blind except the upper part of the field of vision—*i.e.*, the lower portion of the retina alone remained active.

Important aid in localizing the lesion causing the eye symptoms may be obtained by the collateral phenomena—*e.g.*, seat of pain, depressions in skull, abnormal phenomena in areas supplied by other nerves.

Willbrand has very ably drawn a number of inferences from the aggregate of symptoms he has observed in occipital lesions. They are :—

1st, That in homonymous areas of the field of vision in hemianopsia, the light sense cannot be reduced without the perception of form and color being similarly reduced.

2nd, That perception of form and color can be reduced without the light perception suffering.

3rd, That perception of form cannot be affected without color sense also failing.

4th, That color perception only may be affected; the light and form perception escaping.

Upon these data he formulates the following:

1st, The fibres of Gratiolet run to the very periphery of the cortex cerebri of the occipital lobes and cuneus, which form the psycho-optic centre.

2nd, The cortex is divisible into three layers, superimposed, in the outermost of which resides the color sense, in the middle the perception of form, in the innermost the light sense.

Now as the fibres of Gratiolet run to the extreme periphery, the course of any one fibre must be through all these three layers. Now it is evident that a lesion of a fibre occurring in the innermost layer will prevent its functionizing in the middle and outer layers; again, that a lesion in the middle area will prevent its acting in the outermost layer, although the innermost escapes, and, finally, that a lesion may occur in the outermost layer without at all altering the function in the two inner layers.

A very interesting and rare case occurred in my practise last spring:

K., aged 30; incipient symptoms of general paralysis of the insane; vision $\frac{1}{2}$; pupils active; fundus normal. Field free for general vision, but *total color blindness* existed to the left side of both fields of vision—i.e., the right occipital lobes were affected. There was also slight facial paralysis of the left side only noticeable on smiling. Patient complained of occipital headaches.

Another case:

T. S., aged 30; poor vision in both eyes. Nine months ago, while skating, he fell and struck the back of his head. A few days later severe meningitis supervened. Patient was unconscious for several weeks; on regaining consciousness was totally blind. Sight has since been gradually returning. Patient has

still occasional severe occipital headaches. Examination of fundus reveals pallor of both discs with diminished calibre of vessels. Vision : L.E., $\frac{1}{2}$, pupils active ; Jaeger 16.

R.E., fingers ten feet.

Both fields of vision limited all round, the R.E. extremely, and wanting entirely to the left and downwards. The L.E. field not limited to so great an extent as that of the R.E., but is also more limited downwards and to the left. The limitation of the fields, the reaction of the pupils, and the seat of pain point to the occipital lobes as the seat of the lesion.

I have not heard for some time from this patient, but it is likely the vision and field will continue to improve as long as the meningitic exudation keeps on absorbing.

In conclusion, I think these few notes may be of use to the general practitioner in assisting him to localise the site of some cerebral lesions, by studying the fields of vision, which can be done approximately without any special instrument, and also by noting the condition of the pupils. To recapitulate :

1st, Are the same areas of both fields affected ? Then, if so, the lesion must be behind the chiasma, in the tract or occiput ; if same areas are not affected, then the lesion may be in the chiasma, in the nerve, or in the eye itself.

2nd, Is the pupil dilated and inactive ? Then the lesion is anterior to the anterior corpora quadrigemina in the tract if the defect of the field of vision is homonymous. If the pupil is active, at the same time with a homonymous defect of the field of vision, then the lesion is behind the corpora quadrigemina.